Modern Concepts of Cardiovascular Disease

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LIFE EXPECTANCY AND INSURABILITY IN HEART DISEASE*

Medicine is hippocratic in its origins, but in practice does not pursue the Greek ideal of excellence. It is an anachronism that perfection is sought in instrumentation and in understanding of disease mechanisms, but that standards applied to the patient are not set as high. Health norms tend to be expressed in terms of the average, which in many important parameters may be considerably in excess of values optimal for health.

Certain disorders, notably those which predispose to cardiovascular disease, *e.g.*, high levels of blood pressure, weight and serum cholesterol, are so universally prevalent that they skew averages toward disease-producing levels. Their adverse portent may be revealed most clearly in long-term follow-up studies, such as medico-actuarial investigations. These have made it possible to define norms, not only in terms of the average, but, more appropriately, in terms of the optimum or ideal values—limits beyond which there is associated a measurable adverse effect on present or future health and longevity. In addition, long-term insurance studies have contributed to clinical medicine by clarifying the natural history of various disorders, providing a valuable frame of reference for prognosis and for appraisal of the effect of therapeutic measures on the course of disease.

It has been learned, both from insurance and clinical studies, that prognosis and life expectancy in persons with various cardiac abnormalities are vastly better than was formerly considered to be the case. Until recent years, recognition of heart disease too often meant not only a diagnosis but a sentence, foreboding a sedentary and presumably brief future. However, with improved understanding and therapy, insurability has been offered increasingly to persons with practically all types of heart disease. Suitable adjustments in premiums are made based on mortality experience, *i.e.*, the ratio of actual long-term mortality in those with a particular impairment to the over-all mortality of the "standard" population of similar age and sex. In general, substandard insurance contracts are offered where mortality ratios in those with a particular impairment do not exceed 500 per cent; not more than five times as many deaths are expected to occur over a long-term period as occur in the standard population.

Coronary Heart Disease

Accepting the dismal prognostic view which prevailed clinically until recent years, insurance companies, in the past, avoided underwriting persons with known coronary heart disease. However, a sizable and valuable mortality experience has accumulated by virtue of the disability features which were common clauses in former insurance contracts. Disability claims following attacks of myocardial infarction have provided abundant material for follow-up mortality studies. In addition, reflecting a more optimistic clinical climate, experimental underwriting of persons with known coronary disease has been increasingly undertaken. Early experience has begun to emerge from these studies. Although adequate long-term mortality data are still lacking, a swelling number of clinical as well as actuarial studies suggest that life expectancy in selected groups with coronary disease is so much better than was formerly supposed that insurance contracts may be offered on a sound basis without prohibitive expense. Some information is now available concerning the remote prognosis in myocardial infarction, asymptomatic electrocardiographic abnormalities, including bundle branch block and T wave changes, coronary insufficiency

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as revealed by electrocardiographic stress tests, angina pectoris and other less specific chest pain. For details, the reader is referred to a recent review prepared by the authors.[1]

Mortality in the first two years after a coronary attack is of the order of six to seven times normal (mortality ratio of 600 to 700 per cent). Thereafter, it falls progressively with increasing intervals of time after the attack. Other factors have significant bearing on survival after coronary attacks. The milder the acute attack, the more complete the recovery, and the older the age at which it occurs, the more favorable the long-term outlook. In such groups, life expectancy may far exceed the over-all average experience, which in most studies has been that three of five persons surviving acute infarction live a further five years, and one in three survives ten years. Conversely, in persons who have accompanying disorders predisposing to progression of coronary disease, such as diabetes, the long-term outlook is poorer than average.

Mortality ratios are somewhat better in persons with angina pectoris than in those who have had known attacks of myocardial infarction, although in one very extensive study the five and ten year survival rates were not significantly higher. Long-term prognosis is slightly better in females than in males, both in angina pectoris and after coronary attacks.

Asymptomatic electrocardiographic abnormalities also have an adverse effect on survival. Mortality in those with major T wave abnormalities is approximately 3½ times normal, and in those with minor T wave changes it is twice normal. Bundle branch block, at one time considered an ominous finding, is not (in the absence of accompanying physical impairments) associated with any striking increase in mortality ratios and is prognostically less unfavorable than T wave abnormalities. Among those with ischemic electrocardiographic changes after exercise, mortality rates are almost threefold greater than in those with a negative response.

Actuarial investigations, as well as epidemiological studies on the rate of development of coronary heart disease, indicate that the risk of developing overt disease may be multiplied several fold in the presence of various predisposing factors, particularly when multiple factors are present concurrently. Among these are elevated serum cholesterol, obesity, hypertension, diabetes, strong family history of coronary disease, heavy smoking, sustained stressful personality pattern, and arcus senilis.

There are indications that the course of coronary heart disease may be modified and the pattern of survival may begin to approach that of the average population by controlling some of the predisposing factors, and by application of such measures as dietary restriction of fat and prolonged use of anticoagulants.

Persons with coronary heart disease, of whatever type, as groups have an abbreviated life expectancy. The great majority succumb to complications of coronary disease, most commonly to recurrent acute coronary attacks or sudden death and, particularly in older persons, to congestive heart failure. However, clinical and actuarial experience indicate that long-term mortality ratios following attacks of myocardial infarction or other syndromes of coronary heart disease are not prohibitive; insurability may be considered in appropriate circumstances, *e.g.*, at an interval after recovery from acute attack, where functional recovery is good, or where there are no accompanying adverse factors.

Hypertension

Hypertension is a composite of many disorders, but these are rarely differentiated. In questions such as those involving insurability, one must necessarily treat all cases of elevated blood pressure as a statistical group. Blood pressure may be considered abnormal at a level at which it leads to two measurable adverse effects: cardiac hypertrophy and an abbreviated life expectancy. This occurs at or near the generally accepted standard of 140 mm. Hg systolic and 90 mm. Hg diastolic. Standards based on average values are fallacious because they are weighted, particularly in older persons, with the very considerable body of the population with pathological hypertension. Actually, there is no sharp dividing line between hypertension and normal blood pressure. The best mortality is experienced with blood pressures considerably below the average. There is a slowly rising mortality with a rise in both systolic and diastolic levels to the so-called average levels, with an accentuated rise appearing above the 140 systolic and 90 diastolic levels.

With the exception of the rather distinctive changes in malignant hypertension, there are no specific pathological features of hypertensive disease. Arteriosclerosis, arteriolosclerosis, cardiac enlargement, renal changes and all the other morphological complications which may develop in hypertension can and do occur in the absence of hypertensive disease.

In general, mortality experience is increasingly unfavorable with progressive elevation of the blood pressure, although there is great individual variability. Mortality reaches 500 per cent, five times the expected, at levels of about 186 systolic and 106 diastolic pressure. Many features in the underwriting of hypertension, other than the level of the blood pressure itself, are of great importance. Perhaps the most significant is sex. Hypertension is so much more a benign disease in women than it is in men that considerably more latitude can be exercised in underwriting women with elevated blood pressure. The extensive study of Bechgaard, *et al.*,[2] of 1,038 hyper-

tensive outpatients followed for an average of 19 years, disclosed that within this period four fifths of the men, as contrasted with one half of the women, had died. In another study[3] of 290 patients in whom elevated blood pressure was discovered at a mean age of 37 in men and 38 in women, 41 per cent of males and 29 per cent of females died within an average 10 year follow-up. After a 16 year period, 58 per cent of males and 42 per cent of females had died. However, persons with a labile hypertension—those whose diastolic pressures had a tendency to fall to values below 95 mm. Hg—had a death rate approximately the same as that of the general population. Those with stable hypertension (diastolic blood pressure consistently over 95 mm. Hg) had five times the normal death rate both in men and in women. Fifty-five per cent of the total succumbed from a cerebral cause, 24 per cent from cardiac involvement, 12 per cent from renal complications, and 9 per cent from miscellaneous causes.

Age is another consideration of some prognostic importance in hypertension. It has generally been considered that the course of hypertension is more rapid and severe when it is contracted early in life, although this has been questioned. In a recent study, McMichael[4] found that a large group of hypertensive persons in the fifth decade had a mortality about five times greater than that of the standard population. For approximately the same levels of blood pressure, the mortality was three times the expected in the sixth decade and 1½ times the expected mortality in the seventh decade.

Family history is another important consideration in hypertension. In hypertensive applicants where there is a strong familial tendency towards early deaths, mortality rates are higher. Actuarial data indicate that the combination of overweight and hypertension increases mortality ratios, as does the presence of albumin in the urine even where no distinct renal disorder can be found. Particularly important is the associated presence of glycosuria, for both hypertension and diabetes predispose individually to degenerative vascular complications. When the two are associated, the rate of development of such complications as coronary artery disease is tremendously accentuated.

Any detectable cardiac abnormalities, such as murmurs, arrhythmias, and significant tachycardia, increase mortality substantially in hypertensive applicants. X-rays and electrocardiograms are frequently requested in insurance applicants when the amount exceeds $25,000. The electrocardiogram has particular prognostic value. In a study carried out some years ago, the authors found that in those persons with elevated blood pressure whose electrocardiograms were normal, the ratio of actual to expected mortality was 186 per cent. Those who exhibited the voltage pattern of left ventricular hypertrophy showed a mortality experience of 344 per cent, presumably reflecting a long-standing hypertensive background. The two groups were similar with respect to age, build and an average blood pressure of 158/98.

A new consideration in underwriting hypertensive applicants has been introduced by the general employment of newer and more effective therapeutic agents. These include particularly the Rauwolfia alkaloids, notably reserpine, and the diuretics, chlorothiazide and its hydroderivative. An important underwriting problem posed by the use of these and other agents in hypertension is whether any credit can be given for the reduction of blood pressure. Properly, the problem is not merely whether the blood pressure is reduced, but whether ultimate mortality can be improved. This will require many years to answer. At the present time, there is little factual justification for regarding hypertension as a less serious condition because it is demonstrated that the blood pressure can be successfully reduced. As has already been mentioned, prognosis is vastly more favorable in those with labile hypertension in whom the blood pressure falls spontaneously than in persons with stable or diastolic hypertension. The situation is very similar to that in diabetes, where even with the proven value of insulin some question still remains as to how effectively the long-term vascular complications of diabetes have been reduced by adequate insulin therapy. In hypertension, as in diabetes, it is quite clear that vigorous treatment can overcome some of the acute severe forms of the disease, such as a malignant hypertension, and can temporarily extend life expectancy so that the progress of renal complications can be arrested and cardiac enlargement and electrocardiographic abnormalities may be reversed. However, there is not sufficient evidence, at the present time, to prove that long-term degenerative complications are halted, or to warrant a basic change in underwriting procedures which depend on ultimate mortality. The critical question is whether the person with hypertension will remain under a life-long regimen of adequate treatment. Since this is individually indeterminate, it is still necessary to apply over-all mortality experience in underwriting hypertensive applicants. Another important problem posed by the use of drugs, which enters into insurability, is that most of the agents employed in hypertension possess potential toxicity.

Rheumatic Heart Disease

Heart murmurs are among the most common impairments detected on insurance examinations. The vast material accumulated in intercompany experience has provided a basis for several long-term mortality investigations. One of the most important is the 1951 Impairment Study of the

Society of Actuaries, of 625,000 persons with various impairments, insured by 27 companies, covering the years 1935 through 1950. More than 15,000 cases with constant apical systolic murmurs, presumed to be organic, were included in this study. Over the 15 year period of study, the mortality was double that among the control group of standard risks. When a history of rheumatic fever or streptococcal infection was present in those with an apical systolic murmur, the mortality rate was further increased to 2.4 times that of the controls. When cardiac enlargement was found in association with apical systolic murmurs, the mortality was further affected adversely. Among those with slight enlargement, there were over three times as many deaths as in the controls, and when moderate enlargement was present the number of deaths was almost five times that among standard risks. Similar findings were reported in earlier investigations, such as the Joint Medical Impairment Study of 1909 to 1927, and the New York Life Insurance Company Study of 1925 to 1931.

Systolic murmurs over the aortic area similarly produce adverse mortality figures. Even without accompanying hypertrophy, those with constant systolic murmurs, not transmitted, had a mortality almost twice normal, whereas when the murmur was transmitted to the neck, presumably indicating aortic stenosis, the mortality rate was five times normal.

It is apparent that such studies do not represent a pure sampling of mitral insufficiency in the case of apical systolic murmurs, and that the group with aortic systolic murmurs are even less specific of aortic stenosis. However, this vast experience, diagnostically unrefined though it may be, does indicate that although organic heart murmurs may at times be discounted clinically in advising a particular patient, they cannot be disregarded in appraising ultimate prognosis and life expectancy.

No mortality data based on insurance experience is available for mitral stenosis or aortic insufficiency. Persons with diastolic murmurs at the apex or base have generally been considered uninsurable in the past. Doubtless, many with these lesions have been insured unwittingly, for the murmurs of aortic insufficiency and mitral stenosis are easily overlooked. There is an increasing trend to offer substandard insurance contracts, on an experimental basis, to selected applicants with aortic insufficiency who have no other impairment, no evidence of cardiac hypertrophy and a relatively normal pulse pressure, since it is recognized that this lesion is often relatively benign and compatible with prolonged survival. This may be so as well in mitral stenosis, but the over-all prognosis in mitral stenosis is not favorable.

A recent survey by Rowe, Bland, Sprague and White,[5] in Boston, disclosed findings very similar to the prognostic study of Grant,[6] in England, reported 25 years earlier. In both studies, some 250 patients with mitral stenosis were followed for ten years or more (or to death). At the end of ten years, 42 per cent in Grant's series and 40 per cent in Rowe's series had died. In 20 years, 80 per cent in the Boston group had succumbed. Even among patients whose functional status at the time of initial observation is good, the long-range outlook is not favorable. Thus in the extensive follow-up report of Olesen,[7] from Denmark, on 351 patients with mitral stenosis, 30 per cent of those with minimal impairment of cardiac reserve had died within 20 years.

This 30 per cent mortality experience in a 20 year period among persons with mitral stenosis is identical with that observed by Bland and Jones[8] in a follow-up of 1,000 children with rheumatic fever, and is similar to the experience of Wilson and Lubschez.[9] In the latter study, comprising a follow-up of 1,042 children, 25 per cent had died within a 20 year period, and the mortality rate was approximately 3½ times that of the general population. Both in the study of Bland and Jones and that of Wilson and Lubschez, more than three fourths of the deaths were due directly to rheumatic heart disease and an additional 10 per cent to bacterial endocarditis. Among adults, as revealed in the 1951 Impairment Study conducted by the Society of Actuaries, mortality experience following attacks of rheumatic fever is much better than in childhood rheumatic fever studies, such as those of Bland and Jones and of Wilson and Lubschez. The disparity in mortality experience between the childhood and adult groups is due to several factors, among them the inclusion in the adult group of significant numbers of cases of polyarthritis not due to rheumatic fever. In addition, acute rheumatic fever in childhood is more apt to be associated with carditis and recurrences are more frequent among children. Thus, in the Bland and Jones study, two thirds of the 1,000 children had rheumatic heart disease when first observed and slightly more than two thirds developed recurrent attacks of rheumatic fever within ten years.

Such studies indicate that rheumatic fever, and particularly its frequent sequel of mitral stenosis, are relatively grave disorders, although the outlook currently is doubtless better than it was in the period covered by these investigations. Individual persons with mitral stenosis not infrequently survive to advanced years with relatively little disability, but the majority do not fare well; *e.g.*, in Olesen's series the average age at death was 47. A new horizon for patients with mitral stenosis has been opened by the advent of surgical treatment. With the passage of a decade, studies such as those of Ellis[10] on 1,000 cases followed from one to eight years, and Donzelot, *et al.*,[11] on a lesser number, have shown convincingly that life expectancy and clinical status have been

improved by surgery. In Ellis' study, 81 per cent of class III patients survived seven years, and 59 per cent of class IV patients survived eight years. Encouraging as such data are, the situation with respect to insurability is not improved, for the disease is not cured. Surgery is usually carried out in patients whose disease is already advanced or complicated, and long-range mortality is far beyond insurable limits. Another brilliant clinical achievement in rheumatic heart disease, which has had little carry-over into questions of insurance, is the successful treatment of bacterial endocarditis. Even though cure is effected by antibiotics, life expectancy must be judged by the underlying valvular lesion. In the case of bacterial endocarditis of the aortic valve, particular reservation is necessary because congestive heart failure, due to further valvular incompetence, may follow successful treatment of the infection.

Congenital Heart Disease

Numerically, instances of congenital heart disease do not comprise a large group among insurance applicants, but are increasing rapidly because of improved recognition of congenital lesions and successful surgical attack. Where curative surgery has been carried out and there are no residual structural or functional abnormalities, a normal or near-normal life expectancy may be anticipated and such persons are freely insurable after a year or more has elapsed. This is the situation following repair of patent ductus arteriosus and, perhaps with somewhat greater reservation, coarctation of the aorta and interatrial septal defects of the predominant secundum type. In instances as yet uncommon but increasing rapidly, cure has been accomplished with lesions such as interventricular septal defect, primum atrial defects, pulmonic and aortic stenosis and other anomalies, using a direct open heart approach employing a bypass procedure. Certain congenital anomalies, such as dextrocardia and right-sided aortic arch, are of little or no consequence.

Until the relatively recent advent of special techniques, such as cardiac catheterization and angiocardiography, congenital cardiac lesions were not diagnosed with any accuracy, and the commonest congenital lesion encountered in adult life, interatrial septal defect, was not clinically differentiated until 1941. Mannheimer, Ikkos, and Jonsson[12] reported a follow-up in 1957 of 14 cases, in which a diagnosis of ventricular septal defect had been made 20 to 25 years earlier. Only 2 of the 14 proved to have the lesion when re-examined by more modern techniques; 1 had an atrial defect, another tetralogy of Fallot, and 10 of the 14 had no evidence of heart disease whatever! Clinical diagnosis is undoubtedly more accurate now, even without the aid of specialized procedures. In general, few patients with untreated congenital heart lesions, excepting atrial septal defects, survive well into adult life. A proper perspective of the extremely high mortality of congenital cardiac lesions is obtained by observing the fate of children from birth rather than at a later time in life. In the extensive studies of MacMahon and McKeown,[13,14] in Birmingham, England, it was estimated that of children born alive with congenital heart lesions, 20 per cent were dead by the end of the first week, 30 to 40 per cent by the end of the first month, and 60 to 70 per cent had died by the age of 10. Similar mortality trends were recorded by Carlgren,[15] in Gothenburg, Sweden, although the mortality he observed was somewhat lower, with a 61 per cent survival at follow-up from birth to ages varying between 7 to 16 years. Forty-nine of Carlgren's 369 cases were operated upon. The over-all incidence of congenital heart lesions among live births was 6.4 per thousand.

Those who survive to adolescence or adulthood represent the less severe types of congenital heart lesions. Although life expectancy may be good in individual instances, notably atrial septal defect, a conservative attitude concerning insurability is dictated by the relatively limited longevity of most persons with congenital heart disease. It is this very circumstance which has prompted the advocacy of surgical attack on all types of curable lesions as direct open-heart techniques have evolved. The prospect of open-heart surgery itself imposes a not inconsiderable hazard. Consequently, there is a good basis for insurability at normal or near-normal rates only after surgical cure has been effected.

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REFERENCES

1. Gubner, R. S., and Ungerleider, H. E.: Long-term prognosis and insurability in coronary heart disease. Am. Heart J. 58: 436, 1959.
2. Bechgaard, P., Kopp, H., and Nielsen, J.: One thousand hypertensive patients followed from 16-22 years. Acta med. scandinav. Suppl. 312: 175, 1956.
3. Mathisen, H. S., Jensen, D., Löken, E., and Löken, H.: Prognosis in essential hypertension. Am. Heart J. 57: 371, 1959.
4. McMichael, J.: Management of hypertension. Presented at Brit. Med. Assn., July 1957.
5. Rowe, J., Bland, E. F., Sprague, H. B., and White, P. D.:

Course of mitral stenosis without surgery: ten and twenty year perspectives. Proc. Third World Cong. of Cardiology, Brussels, 1958.
6. Grant, R. T.: After-histories for 10 years of a thousand men suffering from heart disease: study in prognosis. Heart 16: 275, 1933.
7. Olesen, K. H.: Mitral Stenosis: Follow-Up of 351 Patients. (Thesis, M.D., Univ. of Copenhagen.) Trans. from Danish by Axel Andersen. Copenhagen, Munksgaard, 1955.
8. Bland, E. F., and Jones, T. D.: Natural history of rheumatic fever: a 20 year perspective. Ann. Int. Med. 37: 1006, 1952.
9. Wilson, M. G., and Lubschez, R.: Longevity in rheumatic fever, based on experience in 1,042 children observed over period of 30 years. J.A.M.A. 138: 794, 1948.
10. Ellis, L. B.: Clinical results in 1,000 cases of mitral stenosis followed 1 to 8 years after valvuloplasty. Proc. Third World Cong. of Cardiology, Brussels, 1958.
11. Donzelot, E., Heim de Balsac, R., Samuel, P., and Beyda, E.: Life expectation of patients with mitral stenosis with and without operation. Brit. Heart J. 19: 555, 1957.
12. Mannheimer, E., Ikkos, D., and Jonsson, B.: Prognosis of isolated ventricular septal defects. Brit. Heart J. 19: 333, 1957.
13. MacMahon, B., McKeown, T., and Record, R. G.: Incidence and life expectation of children with congenital heart disease. Brit. Heart J. 15: 121, 1953.
14. McKeown, T., MacMahon, B., and Parsons, C. G.: Familial evidence of congenital malformation of heart. Brit. Heart J. 15: 273, 1953.
15. Carlgren, L. E.: Incidence of congenital heart disease in children born in Gothenburg 1941-1950. Brit. Heart J. 21: 40, 1959.

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